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Congestive Heart Failure - A Haematological, Biochemical and Cytogenetic Study

N. Meenakshi, K. Sasikala, K.A. Sajeetha Beegam, A.L. Calistus Jude, N. Balachandar, M.Vimala Devi and A. Asia Begum

Division of Human Genetics, Department of Zoology, Bharathiar University,Coimbatore, Tamil Nadu, India

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ABSTRACT Congestive Heart Failure (CHF) is a condition in which a weakened heart cannot pump enough blood to body organs and the blood backs up into certain body tissues. This study investigates the haematological, biochemical and cytogenetic changes in patients with CHF. Significant alterations were observed in leucocyte count and lipid profile, and Chromosomal analysis revealed major aberrations such as deletions, translocations, inversions, satellite formation, mosaicism and deletion of sex chromosome in the Experimental subjects. The results are discussed pertaining to the recent literature.

INTRODUCTION

The heart is one of the vital organs of human body and is prone to many diseases and disorders. Congestive Heart Failure (CHF) is a clinical syndrome characterized by abnormal sodium and water retention resulting from impaired cardiac function and can be caused by many diseases and conditions (Fowler 1947).

In addition to coronary heart disease and multiple or severe heart attacks, risk factors for CHF include alcohol abuse, certain infectious diseases common in underdeveloped countries, congenital heart disease, diabetes, heart valve damage, high blood pressure and cholesterol levels, obesity, inactive lifestyle, smoking and some genetic disorders that lead to conditions known as cardiomyopathies.

Considering the genetic factors, genes contribute to both the cause and the pathogenesis of virtually any abnormality of human physiology and behaviour including disorders of the heart and vascular system (Childs 1995). During the past 10 years a rapidly expanding list of genes that are involved in the etiology of cardiovascular diseases, including hypertrophic and dilated cardiomyophathies, hypertension and atherosclerosis, have been identified (James et al. 1998).

The present study focuses on analyzing biochemical, nutritional, environmental factors and the genetic changes if any in the patients with congestive heart failure prevailing in Coimbatore city so as to enable the patients to get better treatment.

MATERIALS AND METHOD

Samples for the present study were collected from patients comprising of an unrelated heterogeneous group of subjects attending the cardiology department of private medical science and research institute at Coimbatore city with the help of a questionnaire and after getting an informed consent. The subjects were categorized into two groups based on the age as Group I (45 to 54 years) and Group II (55 years and above). The category also includes sex difference (male n = 48 and female n = 33). Equal number of mentally normal and physically healthy subjects were taken as controls. The blood and sera of the experimentals and controls were utilized for analysis of various haematological, biochemical and cytogenetic parameters.

RESULTS

An overall decrease in the mean erythrocyte count was observed in both the groups of male and female subjects. The mean leucocyte count of experimentals of the two groups of male and female subjects showed an elevated trend than that of the Controls. Except Group II female subjects, all the others showed an insignificant variation. The mean haemoglobin level was decreased among Experimental males of Group I and II and also in Group II females. A slight elevation was seen in the Group I females, but the increase was not statistically significant (Table 1). Significant elevations were observed with regard to Triglycerides, Cholesterol and Low Density Lipoproteins (LDL) in the male and

Table 1: Changes in haematological parameters in various groups of male and female subjects with congestive heart failure

S.No.	Particulars	No. of subjects studied	RBC (million cells/ cu. mm)	WBC (thousand cells/ cu. mm)	Haemoglobin (gm%)
	Group I (Male)				
1	Controls	28	5.266 ± 0.1991	8.9871 ± 0.356	15.0428 ± 0.3475
	Experimentals	28	4.1482 ± 0.168	14.642 ± 0.309	13.6964 ± 0.3987
	Group II (Male)				
2	Controls	20	5.148 ± 0.2501	8.3225 ± 0.4324	14.215 ± 0.4203
	Experimentals	20	4.4205 ± 0.2162	11.65 ± 0.4254	13.41 ± 0.4600
	Group I (Female)				
3	Controls	15	4.814 ± 0.3272	5.838 ± 0.4549	9.6346 ± 0.2871
	Experimentals	15	4.2326 ± 0.2822	12.3474 ± 0.733	10.5533 ± 0.2688
	Group II (Female)				
4	Controls	18	4.49 ± 0.2242	6.745 ± 0.381	10.8055 ± 0.3216
	Experimentals	18	4.38 ± 0.2094	12.1361 ± 0.647*	10.3944 ± 0.3005

Group I: 45 - 54 years of age
Group II: 55 years and above

* Value significant at 5 % level
* * Value significant at 1% level

Table 2: Lipid profile and enzyme assay in various groups of male and female subjects with congestive heart failure

S. No.	Particulars	No. of Sub.	Triglycerides (mg/dL)	Cholesterol (mg/dL)	High Density Lipoprotein (mg/dL)	Low Density Lipoprotein (mg/dL)	Mean Value of SGOT (units /L)	Mean Value of SGOT (units /L)
	Group I (Males)							
1	Controls	28	143.07 ± 1.48	209.57 ± 6.83	37.71 ± 0.68	143.24 ± 6.78	25.96 ± 1.52	28.25 ± 0.83
	Experimentals	28	218.5 ± 6.41*	266.93 ± 3.84*	31.71 ± 1.27*	182.44 ± 5.13 **	66.78 ± 4.93**	82.5 ± 1.48**
	Group II (Males)							
2	Controls	20	143.7 ± 1.59	229.2 ± 5.97	39.5 ± 0.96	161.32 ± 6.25	30.45 ± 1.78	27.5 ± 0.98
	Experimentals	20	187.15 ± 10.37*	265.17 ± 2.67*	33.5 ± 1.08	188.87 ± 3.74*	53.9 ± 3.06**	79.55 ± 2.47**
	Group I (Females)							
3	Controls	15	146.26 ± 0.85	245.06 ± 1.43	37.14 ± 0.85	178.69 ± 1.78	31.26 ± 1.41	28.26 ± 0.97
	Experimentals	15	181.2 ± 11.91*	257.75 ± 5.15*	37.86 ± 1.82**	192.82 ± 4.95**	43.06 ± 1.79	90.8 ± 4.95**
	Group II (Females)							
4	Controls	18	144.0 ± 1.02	246.05 ± 0.88	34.1 ± 0.76	183.11 ± 0.72	30.0 ± 1.23	30.11 ± 0.54
	Experimentals	18	155.78 ± 3.47*	259.35 ± 3.09*	42.5 ± 2.53*	192.06 ± 3.84**	44.11 ± 2.25**	97.27 ± 4.50**

Group I: 45 - 54 years of age
Group II: 55 years and above

* Value significant at 5 % level
* * Value significant at 1% level

Table 3: Details of chromosomal aberrations in male and female subjects with congestive heart failure

S.No.	*Case ID*	*Particulars*	*Chromosomal Aberration (Males)*
1	HGCHF2	Group I	46,XY, del (13q-)
2	HGCHF10	Group II	46,XY, inv (9)
3	HGCHF12	Group II	46,XY, t (5p-; 12q+)
4	HGCHF15	Group II	46,XY, inv (9)
5	HGCHF16	Group I	46,XY, 13 s+
6	HGCHF28	Group II	46,XY, del (13q-)
7	HGCHF31	Group I	46,XY/ 46,XY,t (6p- ; 22q+)
8	HGCHF33	Group II	46,XY, t (5p- ; 12q+)
9	HGCHF34	Group I	46,XY, t (14q- ; 22 q+)
10	HGCHF38	Group II	46,XY,del (18p-)
11	HGCHF45	Group II	46,XY,del (13q-)
12	HGCHF47	Group II	46,XY, del (Xq-)
S.No.	*Case ID*	*Particulars*	*Chromosomal Aberration (Females)*
1	HGCHF3	Group II	46,XX, inv (9)
2	HGCHF7	Group I	46,XX / 45, XO/ 46, XX, 22s^{+}
3	HGCHF11	Group I	46,XX, del (7p-)
4	HGCHF19	Group II	46,XX, t (5p- ;21q+)
5	HGCHF20	Group I	46,XX, del (22q-)
6	HGCHF23	Group II	46,XX, del (18q-)
7	HGCHF28	Group II	46,XX, del (15p-)
8	HGCHF32	Group II	46,XX, del (18q-)

female Experimental subjects of Groups I and II High Density Lipoprotein was significantly decreased in male subjects of Group I and II while the level increased in the female subjects of Group I and II. Serum transaminases (Serum glutamic-oxaloacetic transaminase and Serum glutamic-pyruvic transaminase) in all the Experimentals showed an elevation when compared with the Controls and was statistically significant except for the Group I females which showed statistically insignificant results (Table 2). 12 male experimental subjects out of 48 displayed chromosomal aberrations such as deletion, translocation, inversion, satellite formation, mosaicism and deletion of a sex chromosome. Of the 33 female experimentals, 8 of them displayed chromosomal aberrations such as deletion, translocation and mosaicism (Table 3).

DISCUSSION

While major advances in the understanding of pathophysiology of congestive heart failure have resulted in treatments that lead to symptomatic improvement and longer life, congestive heart failure remains a major clinical challenge, both in its early, accurate diagnosis and treatment.

Epidemiological studies have shown that the risk of cardiovascular disease steadily increases with increasing levels of plasma cholesterol and that it is difficult to define a 'healthy' range. In the present study, statistically significant values of elevation were estimated in the levels of triglyceride in both the groups of male and female subjects, which matched the results of various researchers (Ball and Mann 1988).

The High Density Lipoproteins (HDL) are a group of lipoprotein which contain cholesterol and several apolipoproteins with various functions. Rising HDL cholesterol reduces body weight and lowers blood pressure and thereby reduces the risk of cardiovascular diseases. The experimentals of both the groups of male subjects expressed a decreased level of serum HDL when compared with their respective Controls and the Group I subjects showed a significant decrease. The experimentals and Controls of both the Groups of female subjects of the present study displayed a slight increase in HDL values which showed a statistical significance. Familial hypercholesterolaemia is generally characterized by more severely elevated LDL cholesterol (Slack 1969). In the present study the LDL cholesterol level was elevated in both the groups of male and female subjects.

The activity of a number of enzymes in serum

are used as an aid to the evaluation of patients with hepatobiliary diseases. Most liver function tests may be abnormal in congestive heart failure with right ventricular decompensation. (Wilkinson 1970). According to Widmann (1984) in liver disease, serum levels of serum glutamic-oxaloacetic transminase and serum glutamic-pyruvic transaminase end to change in parallel, with congestive heart failure. Myocardial infarction causes in addition, substantial increase in SGOT because this enzyme is abundantly present in the heart muscle. The SGOT and SGPT values are raised in both the Groups of male and female subjects of the present study.

Hundreds of genes are likely to be important in the development and normal physiology of the cardiovascular system. Schinzel (1983) and Borgaonkar (1994) demonstrated that upto 40 per cent of all foetuses with heart defects detected by ultrasonography at 18 to 20 weeks gestation have chromosome aberration. Most forms of aneuploidy and most duplications and deletions of more than a chromosome band are associated with defects of the cardiovascular system. In the present study, deletion in various chromosomes was the major chromosomal aberration in both male and female subjects with CHF apart from other major and minor aberrations.

CONCLUSION

Genetic analysis of a simple blood sample from a child will allow the accurate prediction of the susceptibility of that child to a wide variety of diseases like hypertension, atherosclerosis, etc. This type of genetic susceptibility testing will fundamentally change the practice of medicine. Rather than treating patients only after they develop symptoms of cardiovascular disease it will be possible to identify those patients at a high risk in childhood and intervene to reduce risk factors before the development of disease condition.

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